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ORIGINAL ARTICLE

Pelvic floor electrophysiology patterns associated with faecal incontinence

Hussein Al-Moghazy Sultan [a], Mohammed Hamdy Zahran [b], Ibrahim Khalil Ibrahim [a], Mohammed Abd El-Salam Shehata [c], Gihan Abd El-Lateif El-Tantawi [a,*,1], Emmanuel Kamal Saba [a]

[a] *Physical Medicine, Rheumatology and Rehabilitation Department, Faculty of Medicine, University of Alexandria, Egypt*
[b] *Radiodiagnosis Department, Faculty of Medicine, University of Alexandria, Egypt*
[c] *General Surgery and Colorectal Surgery Department, Faculty of Medicine, University of Alexandria, Egypt*

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KEYWORDS

Faecal incontinence;
Pudendal nerve terminal motor latency;
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Puborectalis electromyography

Abstract *Introduction:* Pelvic floor electrophysiological tests are essential for assessment of patients with faecal incontinence.
Aim: The present study was conducted to determine the patterns of pelvic floor electrophysiology that are associated with faecal incontinence.
Subjects: The present study included 40 patients with faecal incontinence and 20 apparently healthy subjects as a control group.
Methods: All patients were subjected to history taking, clinical examination, proctosigmoidoscopy, anal manometry and electrophysiological studies. Electrophysiological studies included pudendal nerve motor conduction study, pudendo-anal reflex, needle electromyography of the external anal sphincter and puborectalis muscles, pudendal somatosensory evoked potential and tibial somatosensory evoked potential. The control group was subjected to electrophysiological studies which include pudendal nerve motor conduction study, pudendo anal reflex, pudendal somatosensory evoked potential and tibial somatosensory evoked potential.
Results: The most common pelvic floor electrodiagnostic pattern characteristic of faecal incontinence was pudendal neuropathy, abnormal pudendo-anal reflex, denervation of the external anal

Abbreviations: FI, Faecal incontinence; PNTML, pudendal nerve terminal motor latency; PAR, pudendo-anal reflex; SEP, somatosensory evoked potential; EAS, external anal sphincter; PR, puborectalis; EMG, electromyography

* Corresponding author. Address: Department of Physical Medicine, Rheumatology, and Rehabilitation, Faculty of Medicine, Alexandria University, Medaan El-Khartoom Square, Al-Azaritah, Alexandria, Egypt. Tel.: +20 01282402836.
E-mail addresses: drhusseinsultan2011@yahoo.com (H.A.-M. Sultan), mhzahran@link.net (M.H. Zahran), dr_ibrahim_khalil@hotmail.com (I.K. Ibrahim), masalam2000@yahoo.com (M.A.E.-S. Shehata), gihan_younis@yahoo.com (G.A.E.-L. Al-Tantawi), emadaziz55@yahoo.com (E.K. Saba).
[1] Present/permanent address: Department of Physical Medicine, Rheumatology, and Rehabilitation, Faculty of Medicine, Alexandria University, Medaan El-Khartoom Square, Al-Azaritah, Alexandria, Egypt.

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sphincter and puborectalis at rest, incomplete interference pattern of the external anal sphincter and puborectalis at squeezing and cough and a localized defect in the external anal sphincter.
Conclusion: There were characteristic pelvic floor electrodiagnostic patterns for faecal incontinence.

1. Introduction

Faecal incontinence (FI) is an anorectal disorder which is common in the community.[1] There is an accumulation of evidence about the importance of neuromuscular function in maintaining continence. Electrophysiological tests are not a substitute for manometric or radiologic studies, but they are complementary to them.[2] The present study was conducted to determine the patterns of pelvic floor electrophysiology that are associated with FI.

2. Subjects

The present study included 40 patients with FI. They were referred randomly from the Outpatient Clinic of Colorectal Surgery Unit of General Surgery Department, Main University Hospital in the years 2009–2011. The study was explained to the participants and an informed consent was given by each. A control group of 20 apparently healthy subjects free from any anorectal or neurological deficits was included. Patients with idiopathic FI, traumatic FI secondary to surgical trauma, obstetric trauma or other trauma and patients with rectal prolapse were included. Patients with diarrhoea, inflammatory bowel diseases, neurological diseases and overflow incontinence were excluded.

3. Methods

All patients were subjected to:

1. History taking that included demographic data and history of the present condition concerning anorectal symptoms. Past history included vaginal deliveries, episiotomy, previous anorectal disorders/surgery and any previous gynecological disorders/surgery.
2. Clinical examination of the perineum, rectum and vagina was performed to detect the presence of perineal descent. Digital anorectal examination aimed to detect and assess: (i) the presence of any sphincter defects; (ii) any other anorectal pathology as rectocele and rectal prolapse.
3. Proctosigmoidoscopy was done by the use of a flexible sigmoidoscope (Fujinon EC 250 WLS Lower GIT Endoscopy, Germany). Its aim was to detect any concomitant rectal diseases and identify patients with any exclusion criteria.[3]
4. Anal manometry was performed using a Synectics (Stockholm) microcapillary perfusion system using a water perfused catheter (MUI Scientific Pump Perfusion System, Mississauga, Ontario, Canada), applying a stationary pull-through technique.[4] All results were recorded using a computerized recording device (Hardware Smartlab, Sandhill Scientific Inc., USA). The following measures were obtained: mean resting anal pressure, mean squeeze anal pressure, high-pressure zone, functional anal canal length and rectal sensations including threshold sensation, urgency sensation and maximum tolerated sensation.
5. Electrophysiological studies were conducted on a NIHON KOHDEN Neuropack MEB-7102 mobile unit with a two channel evoked potential / EMG measuring system (Nihon Kohden Corp., Tokyo, Japan). The study included the following: (i) Pudendal nerve motor conduction study for the right and the left sides to measure pudendal nerve terminal motor latency (PNTML). It were performed according to the technique described by Roberts[5] (ii) Pudendo-anal reflex (PAR) was performed according to the technique described by Roberts[5] (iii) Needle electromyography (EMG) of the EAS during rest, squeezing, straining and cough, and mapping of the muscle were performed according to the technique described by Roberts[5] (iv) Needle EMG of PR muscle during rest, squeezing, straining and cough were performed according to the technique described by Roberts[5] (v) Pudendal SEP was done for the exclusion of central neurological disorders. It was performed according to the technique described by Podnar[6] (vi) Tibial SEP was done for the exclusion of central neurological disorders. It was performed according to the technique described by Cruccu et al.[7] The control group was subjected to electrophysiological studies which include pudendal nerve motor conduction study, pudendo-anal reflex, pudendal somatosensory evoked potential and tibial somatosensory evoked potential.
6. Statistical analysis of data was done by using the Statistical Package of Social Science (SPSS version 17) software.[8] Descriptive measures (count, frequency, minimum, maximum, mean and standard deviation) as well as analytic measures (t-test and Pearson Chi-square test) were used. Statistical significance was assigned to any P value at $\leqslant 0.05$. Kappa test was performed to determine consistency among different diagnostic tools (Kappa = 1 when there is perfect agreement, Kappa < 0 when there is no agreement). The cut off value of the electrophysiological studies was equal to the mean plus two standard deviations.

4. Results

The present study included 40 patients with FI [20 men (50%) and 20 women (50%)]. Their mean age was 35.4 ± 14.1 years (ranged from 18 to 70 years). The control group consisted of 20 individuals [10 men (50%) and 10 women (50%)]. Their mean age was 39 ± 15.6 years (ranged from 20 to 65 years). There were no statistically significant differences between patients and control group as regards the gender and age ($P = 1.000$).

The most common type of FI was liquid FI (60%). The aetiologies of FI were rectal prolapse (37.5%), traumatic perineal injury (20%), idiopathic FI (17.5%), post-surgical FI (17.5%) and obstetric FI (7.5%). The mean duration of FI was 4.1 ± 4.2 years (ranged from 0.2 to 15 years). Eleven patients (55% of women) had normal vaginal delivery while two patients (10% of women) had episiotomy scar. Nine patients (45% of women) were nulliparous and none delivered by caesarean section. Past history of anal operations was present in 45% of patients in the form of perineal tear repair (17.5%), haemorrhoidectomy (12.5%), rectal prolapse repair surgery (10%) and posterior sphincterotomy (5%). Clinical evidence of a localized defect in the EAS was present in 25% of patients. There was unremarkable proctoscopic examination among FI patients.

The evaluation of the pelvic floor electrophysiological studies was based on reference values in comparison to the control group (Table 1). Results of the electrophysiological studies were compared to the clinical and manometric findings. The pelvic floor electrophysiological abnormalities associated with FI were illustrated in Table 2.

The PNTML was significantly prolonged in FI group compared to control group. There was no statistically significant difference between FI group and control group as regards PAR latency. There were no statistically significant differences between FI group and control group as regards pudendal SEP and tibial SEPs (Table 3). All FI patients had pudendal SEP and tibial SEP parameters within the cut-off points i.e. none of them had abnormal findings.

There were eight different pelvic floor electrophysiological patterns among FI patients according to the pudendal nerve conduction study. All patterns are illustrated in Table 4. The most common patterns were patterns VII, VIII and I.

The anal manometry changes in the anal pressures, anal lengths and anal sensations are shown in Table 5.

Among the 40 patients with FI, there were 20 patients with anal sphincter localized defect by EMG mapping of the EAS. The aetiology of FI in these 20 patients was: six patients with FI secondary to rectal prolapse, seven patients with FI due to traumatic perineal injury, three patients with post-surgical FI, three patients with obstetric FI and one patient with idiopathic FI.

Among the 40 patients with FI, 9 out of 10 patients who had an anal sphincter localized defect by clinical examination were found to have a localized defect by EMG mapping of the EAS. However, patients who had no localized defect in anal sphincter by clinical examination, 11 showed a significant localized defect by EMG mapping of the EAS. There was a fair agreement (Kappa = 0.400, $P = 0.003$) between detection of a localized anal sphincter defect clinically and electromyographically through EMG mapping of the EAS muscle.

Among FI group, mean squeeze pressure was significantly lower among patients with bilateral pudendal neuropathy in comparison with patients with unilateral or no pudendal neuropathy ($\chi^2 = 10.765$, $P = 0.029$). Mean squeeze pressure was significantly lower among patients with decreased recruitment pattern of the EAS muscle at rest (sign of denervation) in comparison with patients with normal recruitment pattern of the EAS muscle at rest ($\chi^2 = 8.714$, $P = 0.013$). Other than these, there were no statistically significant differences between patients with abnormal electrophysiological findings and those with normal findings as regards anal manometry.

Table 1 The determined cut-off points for different electrophysiological studies.

Electrophysiological studies	Determined cut-off point
Mean PNTML (ms)	⩽ 2.28
PAR latency (ms)	⩽ 43.04
Pudendal SEP latency (ms)	⩽ 44.11
Tibial SEP latency (ms)	⩽ 44.84

Table 2 Common pelvic floor electrophysiologic abnormalities associated with FI.

Common pelvic floor electrophysiologic abnormalities	Number of patients ($n = 40$) (%)
Pudendal neuropathy [unilateral / bilateral]	34(85) [5/29] ([12.5/72.5])
Abnormal PAR	19 (47.5)
Signs of EAS denervation	36 (90)
Decreased recruitment of EAS at rest	35 (87.5)
Sign of EAS reinnervation	32 (80)
EAS incomplete interference pattern at squeezing and cough	32 (80)
Anismus of EAS	11 (27.5)
Localized defect in EAS mapping	20 (50)
Abnormal EMG of EAS	39 (97.5)
Signs of PR denervation	27 (67.5)
Decreased recruitment of PR at rest	22 (55)
Signs of PR reinnervation	20 (50)
PR incomplete interference pattern at squeezing and cough	19 (47.5)
Anismus of PR	16 (40)
Abnormal EMG of PR	36 (90)

5. Discussion

Pelvic floor electrophysiological studies are essential for the diagnosis and management of anorectal disorders in the form of FI. They help in the localization of the lesion, determination of the mechanism of injury and assessment of the severity especially when there is a completely normal neurological examination.[2]

In the present study, about 72.5% of FI patients had bilateral pudendal neuropathy and 12.5% had unilateral pudendal neuropathy. The cause of pudendal neuropathy among FI patients was attributed to the susceptibility of the terminal portion of the pudendal nerve to injury as a consequence of rectal descent and labour in the form of traction neuropathy. Patients with anal sphincter defect of any kind as traumatic perineal injury or iatrogenic surgical anal sphincter defect may be associated with pudendal neuropathy. This occurs with a major defect in the EAS. Pudendal neuropathy appears when its effector muscle is severely affected.[9] This is important from the management point of view. The clinical significance of bilateral pudendal neuropathy is its association with poor surgical repair outcome.[10]

Table 3 Comparison of PNTML, PAR latency, pudendal and tibial SEP between FI and control groups.

Electrophysiological studies	FI group mean ± SD	Control group mean ± SD	*t*	*P*
Rt side PNTML (ms)	3.16 ± 2.04	1.9 ± 0.24	2.519	0.015*
Lt side PNTML (ms)	3.31 ± 2.14	1.86 ± 0.16	2.577	0.013*
PAR latency (ms)	39.24 ± 17.55	37.70 ± 2.67	0.389	0.699
Pudendal SEP P40 latency (ms)	40.60 ± 1.90	41.84 ± 2.28	1.066	0.291
Tibial SEP P40 latency (ms)	39.02 ± 2.32	40.07 ± 2.94	0.935	0.354

SD = Standard deviation.
* *P* is significant at $\leqslant 0.05$.

Table 4 Patterns of pelvic floor electrophysiological abnormalities among FI patients.

Pelvic floor electrodiagnostic patterns depends on pudendal nerves function	I	II	III	IV	V	VI	VII	VIII
Pudendal nerve function (normal/unilateral pudendal neuropathy/bilateral pudendal neuropathy)	Normal	Unilateral	Unilateral	Bilateral	Bilateral	Bilateral	Bilateral	Bilateral
Abnormal PAR	3		1		1	2	5	6
EMG of EAS								
Denervation	6	2	3		4	3	11	10
Reinnervation	2	2	3		4		11	10
Incomplete interference	4	2	2		4	3	9	9
Anismus	3	1	1				5	1
Localized defect	3	1	2		3	1		10
EMG of PR								
Denervation	5		2			3	11	10
Reinnervation	1		2				7	10
Incomplete interference	2		1			1	7	9
Anismus	3	1	2				5	3
Total number of patients	6	2	3	1	4	3	11	10

Number: number of patients.

Table 5 Descriptive data of parameters of anal manometry among FI groups.

Anal manometry changes	FI group (mean ± SD)
Resting pressure (mmHg)	43.50 ± 25.70
Squeeze pressure (mmHg)	91.63 ± 50.03
Functional anal canal length (cm)	2.16 ± 1.48
High pressure zone length (cm)	0.97 ± 0.89
Threshold sensation (ml H_2O)	23.13 ± 12.49
Urgency sensation (ml H_2O)	42.12 ± 13.05
Maximum tolerated sensation (ml H_2O)	133.50 ± 19.58

SD = Standard deviation.

The present study is in accordance with Roig et al.[11] who reported that pudendal neuropathy was present in 69.8% of FI patients (20.8% unilateral pudendal neuropathy and 49.2% bilateral pudendal neuropathy).

Vaccaro et al.[12] found that in FI patients, bilateral pudendal neuropathy was present in 21.5% and unilateral pudendal neuropathy in 15.7%. Ricciardi et al.[13] reported that 28% of FI patients had unilateral pudendal neuropathy while 12% had bilateral pudendal neuropathy. These studies are not in accordance with the current study. This could be due to several causes as the difference in aetiology of FI, as well as, pudendal nerve conduction study measures only the fastest conducting fibres along the nerve bundle; it could overlook nerve damage if even a small number of fast conducting fibres are conducting normally. Also, it could be due to the more severe advanced lesion and late medical consultation among the Egyptian patients.

In the present study, patients with FI had prolonged PAR in 47.5% of cases. This could be explained by the presence of an abnormal reflex arc due to the presence of pudendal neuropathy. If few intact pudendal nerve axons are present, it is sufficient to produce a PAR reflex of normal latency. The PAR has a wide physiological range of latencies and so the delayed PAR latency by few milliseconds might not show itself as a significant change in PAR latency. Fowler et al.[14] reported that the latency of PAR lacks sensitivity in establishing minor to moderate lesions of the pudendal nerve or sacral nerve roots as these lesions are axonal. Because the assessment of PAR is based on the conduction (and not the amplitude), they are not sensitive to incomplete lesions, whether demyelinating or axonal. Thus, a normal response does not exclude a lesion.[5] The present study is in accordance with Fitzpatrick et al.[15] who reported that 50% of patients with obstetric FI had an abnormal PAR. The present study is not in accordance with Varma et al.[16] who showed that all neurogenic FI patients had an abnormal PAR. The difference between the results of the current study and Varma et al.[16] can be due to the difference in the aetiology of FI as all their patients were neurogenic FI.

In this work, the mean pudendal SEP and tibial SEP latencies of FI patients and healthy control did not differ in a significant fashion. This indicated the absence of any neurological deficit either central or peripheral among the patients. Delodovici et al.[17] showed that abnormal pudendal SEP was found only in patients with abnormal neurological examination.

In the present work, abnormal EMG of the EAS was present in 97.5% of FI patients. The presence of denervation in the EAS EMG is an indicator of ongoing neurogenic injury to the EAS. This denervation could be due to the injury of the terminal part of the pudendal nerve as a consequence of perineal descent during delivery. This finding supports the concept that a main finding of FI is denervation of the EAS muscle due to damage of its innervation. This damage of the EAS could be prevented by an early diagnosis and treatment of rectal prolapse and a proper anal surgery to prevent anal sphincter injury. Pelvic floor exercises following labour and following perineal surgery result in prevention of further denervation of the EAS muscle. Complex repetitive discharge was not detected in any patient. This can be due to the exclusion of patients with neurological lesions or disorders. The included patients in the current study had only local anorectal lesions. The complex repetitive discharge occurs in neurologic lesions and chronic neuropathic disorders.[18] The presence of signs of reinnervation in the form of polyphasic MUAPs indicates the presence of chronic reinnervation following denervation. It also indicates that the axonal injury was not recovered completely. Among FI patients, the presence of reinnervation is an indicator of potential recovery by treatment of the cause and biofeedback therapy. The incomplete interference pattern at squeeze and cough among FI patients reflects the main problem of FI which is a defect in the function of the EAS in continence control. The presence of anismus in the EAS of FI patients could represent a compensatory phenomenon to prevent faecal soilage or the presence of obstructed defecation in those FI patients before the development of FI.

In the present study, EMG mapping of the EAS among FI patients revealed the presence of a localized defect in 50% of the patients. Clinical examination revealed a localized defect in only 25% of the patients. This can point to the ability of EMG in assessing the muscle integrity.

An important point to observe is that not all patients with traumatic FI had a localized defect in EMG mapping of the EAS muscle. This can be explained by injury to the internal anal sphincter muscle at the sites of pedicle excision or due to loss of the mucosal cushions in haemorrhoidectomy operation.[19] Moreover, cases with a localized defect in EMG mapping of the EAS muscle included patients with idiopathic FI (1 patient) and patients with FI secondary to rectal prolapse (six patients). It was reported that idiopathic FI and FI secondary to rectal prolapse can be associated with a localized defect in EAS mapping.[20–22] It can be due to an unrecognized anal sphincter defect which took place during normal vaginal delivery without obstetric FI among multigravid women as reported by Fundinger et al.[21] The long time lapse between delivery and onset of FI excludes an obstetric FI. Woods et al.[22] studied patients with rectal prolapse and FI who had undergone endosonographic imaging of the anal sphincter. They found that 15 of 21 patients had an abnormality in the anal sphincter complex and concluded that anal sphincter defects may play a role in persistent incontinence following repair of the prolapse.[22]

In the present work, abnormal EMG of PR was present in 90% of FI patients. The EMG abnormalities of PR muscle were similar to those abnormalities of the EAS muscle. In spite of the different motor supply of PR muscle, the explanation of its abnormalities was the same as those of the EAS muscle.

Cheong et al.[23] underwent an electrodiagnostic evaluation of FI patients. The EMG abnormalities of the EAS were present in 46% of patients. There were denervation potentials in 8% of patients and reduced recruitment at rest in 62% of patients. Incomplete interference pattern at squeeze and cough was present in 55.5% of patients. Reinnervation in the form of polyphasic motor units was present in 32%. Anismus was present in 19%. There was a localized defect in EAS mapping in 37%. The difference between this study and the current study can be due to the difference in the frequency of different FI aetiologies.

Snooks et al.[24] studied the innervation of PR and the EAS in FI and rectal prolapse patients. They reported pudendal neuropathy in 68% and reinnervation of PR in 60% of patients while reinnervation of the EAS was present in 75% of patients. The data showed that the different innervations of the PR and EAS are both damaged in patients with FI.

Bartolo et al.[25] reported that FI patients had a significant neuropathic damage to the EAS and PR muscles compared with controls. The findings suggest that partial denervation of the EAS can occur independently of partial denervation of the PR but if severe changes are present in both muscles, the patient is likely to be incontinent. This study is in accordance with the current study.

The electrophysiological findings indicate that nearly all FI patients have one or more neuromuscular defects which contribute to the process of incontinence. This has a role in the decision of the appropriate therapy whether conservative or surgical therapy. Intact or minimal damage is a prerequisite for successful surgical repair treatment.[26]

Most common pelvic floor electrophysiological pattern among FI patients was pattern VII. This occurred in 27.5% of FI patients. This represents a sort of mixed pudendal neuropathy (a mixture of axonal and demyelinating lesion). The axonal component leads to the denervation and reinnervation in the EAS muscle. It is a traction pudendal neuropathy by rectal descent in rectal prolapse or a stretch injury to the pudendal nerve by pelvic floor descent that occurs with chronic straining or previous difficult vaginal delivery. The PR muscle was also affected by denervation and reinnervation. This could be explained by stretching upon the motor nerve supply of PR which arises directly from the sacral plexus in the case of rectal descent in rectal prolapse or direct injury of PR in surgical procedures.

Pattern VIII is similar to pattern VII with the presence of a localized defect in the EAS muscle and occurred in 25% of FI patients. This could be explained by the same explanation as in the previous pattern in addition, a localized defect could be secondary to the perineal injury whether obstetric, traumatic or iatrogenic, or secondary to chronic stretch of the EAS during rectal prolapse descent. The presence of a localized defect in the EAS muscle compromises the role of the EAS in maintaining continence. It has an important role in the decision of the appropriate surgical repair of FI.[26]

The next common pattern was pattern I. This occurred in 15% of FI patients. The abnormal PAR with intact pudendal nerve indicates a proximal pudendal demyelinating lesion but the presence of denervation and reinnervation in the EAS indicates the presence of secondary mild degree of the axonal lesion. The presence of abnormalities in the PR is due to stretching upon the motor nerve supply of the PR which arises from the sacral plexus. There were other less common patterns.

The current study is in agreement with Fitzpatrick et al.[15] Fitzpatrick et al.[15] reported the presence of three main patterns of abnormal pelvic floor electrophysiological studies among obstetric FI patients according to the abnormalities of the pudendal nerve function. The first pattern was demyelinating pudendal neuropathy with an abnormal PAR and normal EAS EMG. The second pattern was axonal pudendal neuropathy with normal PAR and abnormal EAS EMG in the form of denervation with reinnervation. The last pattern was a mixed demyelinating and axonal pudendal neuropathy with abnormal PAR and EAS EMG.

Wexner et al.[27] reported the pelvic floor electrophysiological abnormalities among FI patients. The EMG abnormalities of the EAS were present in 58% of patients. There were denervation potentials in 10% of patients and an incomplete interference pattern at squeeze and cough in 58% of patients. Reinnervation in the form of polyphasic motor units was present in 40%. Anismus was present in 22%. Pudendal neuropathy was present in 35% of patients. The current study is not in accordance with Wexner et al.[27] The difference between this study and the current study can be due to the difference in the frequency of different FI aetiologies.

In FI patients, the different electrophysiological patterns represent a continuous spectrum of changes that extend from a normal pudendal nerve to bilateral pudendal neuropathy with a neurogenic lesion affecting EAS and PR muscles. Different patterns represent a stage in the spectrum of electrophysiological changes that took place in FI. These changes depend on many factors as the aetiology and severity of the lesion causing FI. The severest pattern in FI occured when both the EAS and PR lost their continence functions by their neurogenic damage.

Among FI patients, mean squeeze pressure was significantly low among patients with bilateral pudendal neuropathy in comparison with patients with unilateral pudendal neuropathy or normal pudendal nerve. This is due to the weakness of both halves of the EAS in the presence of bilateral pudendal neuropathy. Mean squeeze pressure was significantly low among patients with decreased recruitment pattern of the EAS muscle at rest in comparison with patients with normal recruitment pattern of the EAS muscle at rest. This means that the EAS muscle's function is subnormal as reflected by the mean squeeze pressure. This needs biofeedback training to enhance the control of weak muscles to perform a better function. The muscle tissue defect itself in traumatic causes of FI as obstetric FI causes mechanical alteration in the mechanism of muscle contraction leading to apparent weakness which could be completely corrected after surgical repair of the muscle. Cheong et al.[23] reported that among FI patients, a reduction in the mean squeeze pressures was associated with an incomplete interference pattern of EAS at squeeze and cough. They reported that the presence of either unilateral or bilateral pudendal neuropathy was not directly associated with any reduction in the squeeze pressure. These are not in accordance with Cheong et al.[23] This can be explained by the difference in the frequency of different FI aetiologies.

6. Conclusions

There are characteristic different pelvic floor electrophysiologic patterns associated with FI. Pelvic floor electrophysiologic changes are a continuous spectrum of changes which vary from minimal changes till severe neuromuscular abnormalities. The most common pelvic floor electrodiagnostic findings of FI are pudendal neuropathy, abnormal PAR, denervation of the EAS and PR, incomplete interference pattern of the EAS and PR at squeezing and cough with or without a localized defect in EAS mapping. Electromyographic mapping of the EAS muscle is a good tool for detection of a localized defect in it.

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